REMARKS

Entry of the foregoing amendment is requested.

Applicants do NOT agree with the statements at point 2 regarding priority.

Example 21 of the current application teaches the stimulation of STAT3. The example is identical to an example in 09/354,243, filed on July 16, 1999. Hence, the priority document 09/354,243 shows all that is claimed, and applicants are entitled to their priority claim.

Should the Examiner disagree, then the Examiner is called upon to explain why commonality of information in a specification does not provide the requisite evidence to support priority.

With respect to the second half of point 2, the inventors did not rename the molecule. Rather, the relevant administrative authorities have. Please see http://www.gene.ucl.ac.uk/cgi-bin/nomenclature/get-data.pl?hgnc_id=14900. A copy of this page is attached.

With respect to points 3, 4, 5, and 6, all relevant corrections have been made.

Applicants will return to points 7-10 presently, but first turn to point 11.

Applicants reduced their invention to practice prior to Ebner, as is shown by the attached Declaration executed by the two co-inventors. Specifically, the inventors verify that the molecule IL-TIF/IL-21 was used to stimulate STAT-3 in both murine and human form. All that a Declaration of this type needs to show is the successful reduction to practice of what is claimed. Applicants have done so. The rejection should be withdrawn.

Turning now to points 7 and 8, the Examiner alleges that the specification is only enabling for *in vitro* stimulation of STAT3 and STAT1 in a hepatoma cell, with specific human sequences and *in vitro* stimulation of mesongial, neuronal, melanoma, and hepatoma cells, using specific murine sequences.

Applicants traverse. The claims require that the stimulated cells be those capable of expressing STAT3. As the Examiner admits, applicants have shown that both human

and murine IL-TIF/IL-21 stimulated STAT3.

Applicants admitted, candidly, in their specification that not all cells do in fact produce STAT-3 when stimulated; however, the claim language is such that not all cells are embraced by the claims. Hence, the issue becomes: would it require undue experimentation to identify relevant cells.

The Examiner then argues that, notwithstanding the provision of five species of TIF, this is insufficient to support the claims. Specifically, the Examiner states that reasonable correlation must exist between claims and enablement.

A specification is presumed to be enabled. It is the Examiner's burden to show that the enablement requirement has not been satisfied. If the Examiner does not, then the specification and claims are enabled.

The Examiner first points to the argument that TIFβ and TIF-α respond differently to IL-9. The Examiner points to examples 12-14 in support.

Examples 12-14 do not discuss any difference between TIF-α and β in the targeted cells. A discussion of TIF-α does not mean TIF-β performs differently. Rather that just making unsupported assertions, the Examiner is called upon to explain why examples 12-14 support his position.

For example, the Examiner points out that STAT3 and IL-TIF/IL-21 are not a ligand receptor binding pair.

How is this relevant?

With respect to the non-prior art citation of Dumoutier, the Examiner misinterprets the paper. Failure to upregulate *in vivo* simply reflects possible need for preactivation by stimuli, such as antigenic or inflammatory stimuli.

Notwithstanding this, the fact remains that STAT3 was stimulated. The Examiner cannot ignore this.

The Examiner then presents an argument at page 5 of the action that is impossible to follow. There is a series of unrelated comments on the technology, which are not linked into an argument to which a response can be directed. While applicants acknowledge the comments, they cannot address them in the absence of clarification.

With respect to the reliance in non-prior art Skolnick, it is again pointed out that the experiments do show that STAT-3 was stimulated, regardless of the source of IL-TIF/IL-21.

With respect to the arguments which then follow, again these amount to unconnected statements regarding the technology generally, and cannot be addressed in the absence of any linking argument.

Allowance of this applicant is believed proper and is urged.

Respectfully submitted,

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~~ISOLATED NUCLEIC ACID MOLECULES WHICH ENCODE T CELL INDUCIBLE FACTORS, OR INTERLEUKIN-21, THE PROTEINS ENCODED, AND USES THEREOF~~

METHODS FOR STIMULATING STAT3 TRANSCRIPTION FACTOR WITH IL-TIF/IL-21

Revised Section of Page 1, lines 2-5

This application is a continuation in part of Serial No. 09/419,568, filed October 18, 1999, now U.S. Patent No. 6,331,613, which is a continuation in part of Serial No. 09/354,243, filed on July 16, 1999, now U.S. Patent No. 6,359,117, which in turn is a continuation in part of Serial No. 09/178,973, filed October 26, 1998, now U.S. Patent No. 6,274,710. All of these applications are incorporated by reference in their entirety.

Revised Section of page 16, lines 15-19

Spleen cells from 10–12 week old ~~Balb/c~~ BALB/c mice were cultured for 24 hours in control medium or the control medium supplemented with 20μg/ml of LPS (which activates B lymphocytes and macrophages), or ConA (which activates T cells), or ConA plus 1% of a blocking antiserum against murine IL-9, with β actin being used as a control. Purification of RNA, RT-PCR analysis were carried out as described supra.

Revised Abstract page 46

ABSTRACT OF THE DISCLOSURE

~~The invention involves isolation of nucleic acid molecules, the expression of which are upregulated by interleukin-9. The amino acid sequences of the proteins which correspond to the nucleic acid molecules show some structural features of cytokines. In addition to the nucleic acid molecules and the proteins, various uses of the molecules are disclosed. The molecules are referred to as T cell inducible factors. The molecules are implicated in activation of STAT molecules, acute phase proteins, and inflammation.~~

The invention relates to methods for stimulating expression of STAT transcription factors, including STAT3, by contacting cells capable of expressing STAT transcription factors with the molecule known, in the alternative, as IL-TIF and IL-21.